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*Case Report*

# Management of Intractable Steroid Induced Ocular Hypertension Following Posterior Subtenons Triamcinolone Acetonide Injection: A Case Report

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## ABSTRACT

Posterior sub-Tenon's (PST) injection of steroid has been used for the treatment of uveitis, cystoid macular edema and choroidal neovascularization. It allows high concentration of the drug to be delivered to the posterior segment of the eye via transscleral route with minimal systemic side effects. Triamcinolone acetonide is an aqueous suspension and it maintains a prolonged intraocular steroid concentration. Steroid-induced ocular hypertension is a complication often seen in patients treated with periocular steroid injections. We report a case of a twelve year old girl who was diagnosed with pars planitis in both eyes. As the inflammation could not be controlled with topical and oral steroids, she was given posterior subtenons triamcinolone acetonide injection. This led to rise in intraocular pressure (IOP) in both eyes. Fundus examination revealed normal optic nerve head in both eyes. The IOP in LE remained normal with anti glaucoma drugs, however in RE it could not be brought under control even with maximum topical and oral anti glaucoma medications. Thus PST implant in the RE was removed 4 months after its injection. The anti glaucoma medications were continued for 3 more months. IOP in RE came under control a month later. Surgical excision of PST should be considered as primary treatment in cases where IOP is not controlled despite maximum medical therapy. Patients with family history of glaucoma, primary open angle glaucoma, diabetes, connective tissue disorders and high myopia are at risk of developing steroid induced ocular hypertension.

***Key words:*** Posterior subtenons triamcinolone acetonide , Steroid-induced ocular hypertension.

## INTRODUCTION

Periocular injection of corticosteroids for the treatment of uveitis was originally described by Nozik in 1972. [1] Posterior sub-Tenon's (PST) injection of steroid has been used for the treatment of uveitis, cystoid macular edema and choroidal neovascularization. PST allows high concentration of the drug to be delivered to the posterior segment of the eye via transscleral route with minimal systemic side effects. Triamcinolone acetonide is an aqueous suspension and it maintains a prolonged intraocular steroid concentration. Steroid-induced ocular hypertension (SIOH) is a complication often seen in patients treated with periocular steroid injections. A periocular injection of steroids was reported to be the more likely to induce an Intra Ocular Pressure (IOP) rise than the administration of other types of corticosteroids. [2] IOP can increase to high levels and if untreated can cause optic nerve damage eventually. [3,4] Patients with family history of glaucoma, history of primary open angle glaucoma, diabetes, connective tissue disorders and high myopia are at risk of developing steroid induced ocular

hypertension. Young age, females and patients with high baseline IOP are also susceptible for the same. Corticosteroids are believed to decrease outflow by inhibiting degradation of extracellular matrix material in the trabecular meshwork, leading to aggregation of an excessive amount of the material within the outflow channels and a subsequent increase in outflow resistance. [5] The clinical features of corticosteroid glaucoma are similar to those of primary open angle glaucoma, with the exception that patients with corticosteroid-responsive glaucoma have a history of significant corticosteroid use.

## CASE REPORT

12 year old girl presented to us with complaints of redness and pain in both the eyes since 2 weeks. On examination her uncorrected visual acuity was 20/20 and intraocular pressure was 10 mm Hg in both eyes. On anterior segment examination she was found to have granulomatous keratic precipitates, grade 2 cells and flare in both eyes. Her fund ii showed pars plana exudates. She was diagnosed with intermediate uveitis secondary to sarcoidosis. She was started on topical steroids, cycloplegics and systemic steroids, which failed to control the inflammation. Thus 9 months after presentation, posterior subtenons triamcinolone acetonide (4o mg/ml) was injected in the left eye. 10 days post injection IOP in LE was found to be 25 mm Hg and 17 mmHg in RE. She was started on brimonidine tartrate and timolol combination in the LE following which the IOP became normal. However, 2 months later there was an IOP spike again and dorzolamide was added. 12 months after presentation posterior subtenons triamcinolone acetonide was given in RE for non healing uveitis (Figure 1). Post PST injection, anti glaucoma medications were started for the right eye. The IOP in LE remained normal with anti glaucoma drugs, however in RE it became 42 mm Hg, 3 months post PST injection. This could not be brought under control even with maximum topical anti glaucoma medications and oral acetazolamide tablets, leading to steroid induced ocular hypertension. The vision remained 20/20 and optic discs of both eyes were healthy .The PST implant in the RE was removed 4 months after its injection due to intractable ocular hypertension (Figure 2, 3). IOP in RE normalized a month later. The anti glaucoma medications were continued in both eyes for 3 more months. After 2 years of follow up, the IOP in both eyes was normal with healthy optic discs without any anti glaucoma medications.

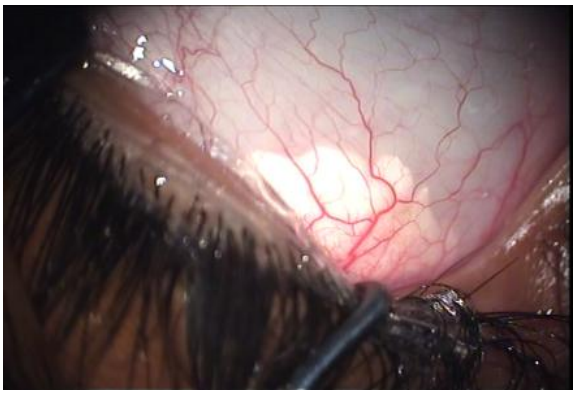

**Figure 1: Posterior subtenons triamcinolone acetonide**

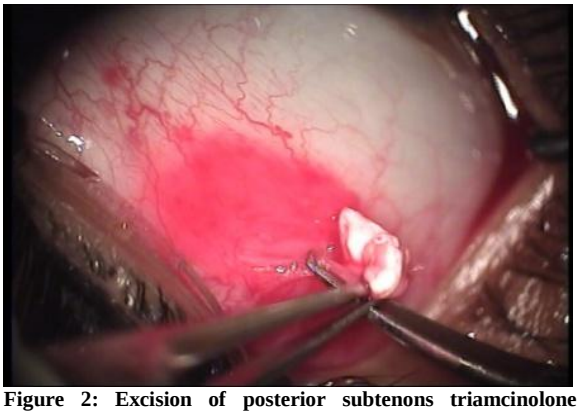

**Figure 2: Excision of posterior subtenons triamcinolone acetonide**

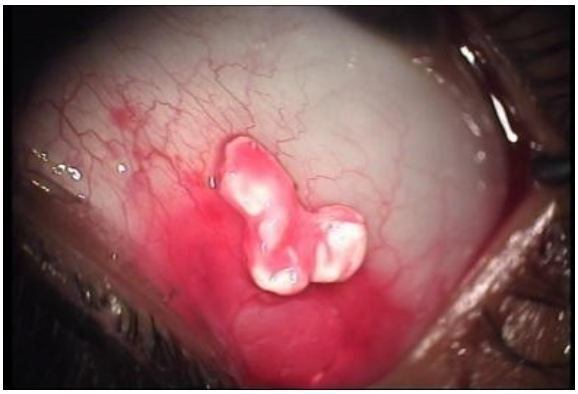

**Figure 3: Excision of posterior subtenons triamcinolone acetonide**

## DISCUSSION

There are varying causes of uveitis associated secondary glaucoma as well as uveitis associated ocular hypertension (OHT), steroids being one of them. Increased IOP (> 21 mm Hg) with optic disc and field changes in uveitis is known as uveitis associated secondary glaucoma and increased IOP with normal optic disc and visual fields is known as ocular hypertension. Takauki et al. [6] reported ocular hypertension in 25.4 % of uveitic patients out of which 45.6 % were steroid induced ocular hypertension. Takahashi et al. [7] reported that steroid-induced glaucoma occurred in 8.9% of cases of uveitis-associated secondary glaucoma. The clinical features of steroid-induced glaucoma / ocular hypertension resemble those of chronic open-angle glaucoma, with a normally appearing anterior chamber angle and the absence of symptoms.

Risk factors for ocular hypertension following subtenon's or inravitreal triamcinolone acetonide were found to be younger age and females. [8] Another risk factors are steroid responsiveness, open angle glaucoma, connective tissue disorders, diabetes mellitus [9] and family history of glaucoma.

he mechanisms responsible for IOP elevation include an excess accumulation of glycosaminoglycans in the aqueous outflow system, suppression of the phagocytic activity of trabecular endothelial cells, and inhibition of the synthesis of PGE2 and PGF2 on glycosaminoglycans of human trabecular meshwork in perfusion organ culture. [10,11]

In the study by Jea et al. , [12] 19 out of 43 eyes (44.2%) in which posterior subtenons triamcinolone acetonide was injected , demonstrated an IOP increase of 5 mmHg or greater. 15 eyes (34.9%) were found to have an IOP of 22 mmHg or higher, and they were treated with IOP-lowering eye drops. The elevated IOP was successfully controlled with IOP lowering eye drops in most patients, except for 2 eyes (4.7%) that had to undergo a trabeculectomy. Most of the topical anti-glaucoma medications were successfully discontinued 12 months postoperatively.

Bui Quoc et al. [13] reported that out of 61, 13 (21.3%) of their patients had increased IOP after a posterior sub-tenon's triamcinolone acetonide injection for noninfectious posterior uveitis and were treated with anti glaucomatous medications. However, the treatment with anti glaucoma medications was unsuccessful in 3 of these cases and surgical excision of periocular corticosteroid deposit was required.

Iwao et al. [14] reported that an IOP of 24 mm Hg or higher was observed in 26 (22.6%) of the 115 eyes within 12 months of posterior sub-Tenon injections of TA, which appeared to be significantly correlated with young age. In total, 23 eyes were treated with anti glaucoma medications to control elevated IOP (24 mm Hg or higher). Trabeculectomy was performed in 1 case where medications failed to correct elevated IOP.

In a study by Yamamoto et al [8] IOP elevation of 5 mm Hg or greater developed in 34.1% of the patients during the sixth month of follow-up after intravitreal or posterior sub- Tenon injections. The mean IOP was found to be increased significantly at the first month, third month, and sixth month follow-up after posterior sub-tenons injection. All eyes that developed steroid-induced glaucoma, except 2, were treated effectively by anti glaucoma agents. Two eyes, which had received posterior sub-Tenon injections for the treatment of diabetic retinopathy, required trabeculectomy.

In a study by Mehmet et al, [15] 18 eyes of 14 subjects with increased IOP within 6 months of sub-Tenon TA injection who did not respond to medical anti glaucomatous treatment were included. IOP levels decreased significantly after the removal of the deposits (mean 15.3 mm Hg [SD 2.1]) ($p < 0.001$) Within 6 months of follow-up, all glaucoma medications were stopped in 9 subjects without further IOP

increase, whereas IOP control in 5 subjects necessitated using glaucoma medications.

In a study by Chan et al, [16] data from 8 eyes of 7 patients undergoing excision of subtenon TA plaques for uncontrolled ocular hypertension after PSTA injection were retrospectively collected. All cases achieved IOP normalization within a mean of 2.5±1.9 days (range, 1 to 5 d) after subtenon TA removal. Fewer kinds of anti glaucoma agents used before subtenon TA removal was associated with greater IOP lowering on postoperative day 1 (P=0.01) and more rapid return to normal IOP (P=0.01).

In our patient too, the PST implant had to be removed 4 months after its injection due to intractable ocular hypertension. IOP thereby normalized a month later. The anti glaucoma medications were continued in the eye for 3 more months and then discontinued.

## CONCLUSION

Surgical excision of PST should be considered as primary treatment in cases where IOP is not controlled despite maximum medical therapy. IOP in cases of periocular steroid injections can increase as late as 6 months leading to refractory glaucoma, thus careful IOP monitoring at every follow up is needed. Special care should be taken in case of younger subjects , females, patients having prior glaucoma or history of steroid responsiveness , those with family history of glaucoma , diabetes and connective tissue disorders.

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